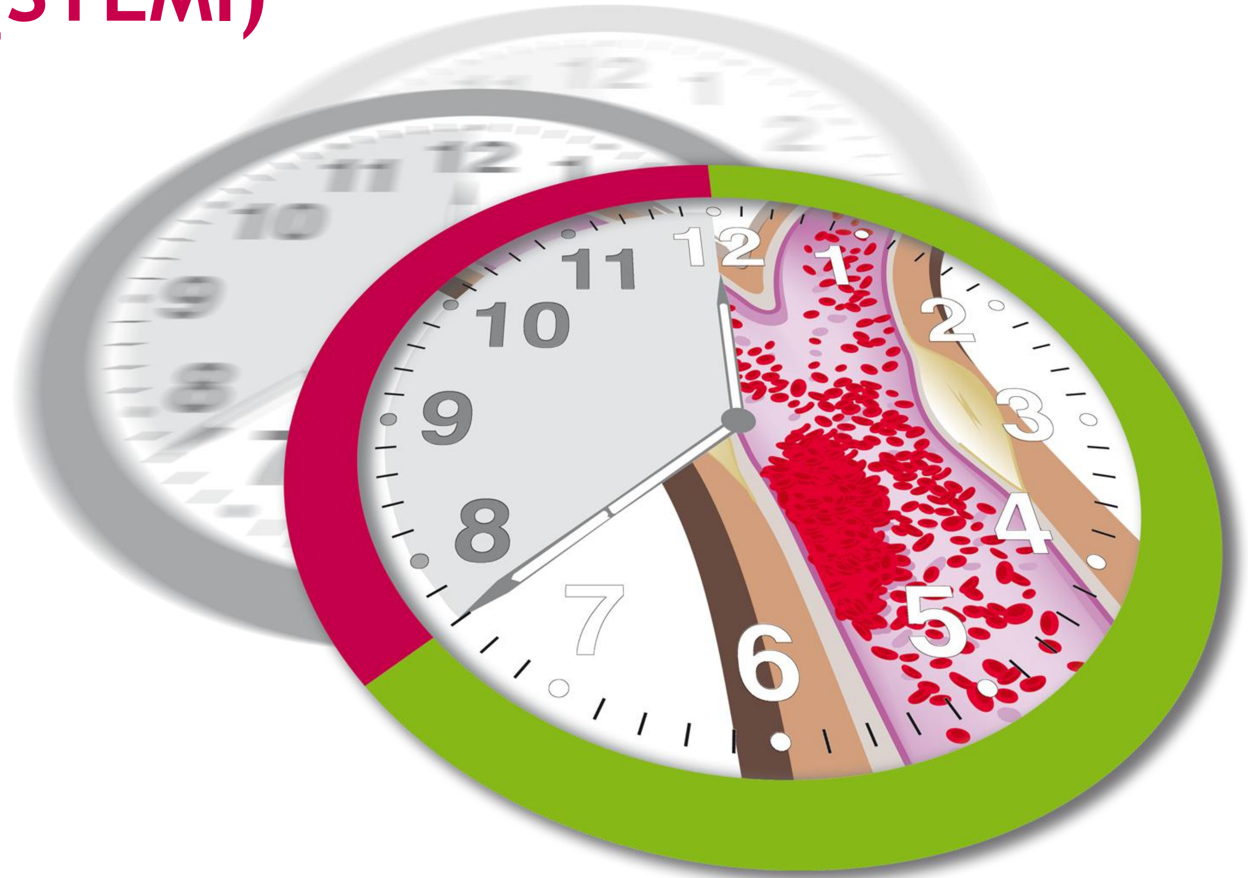
ST-elevation myocardial infarction (STEMI)

The basics

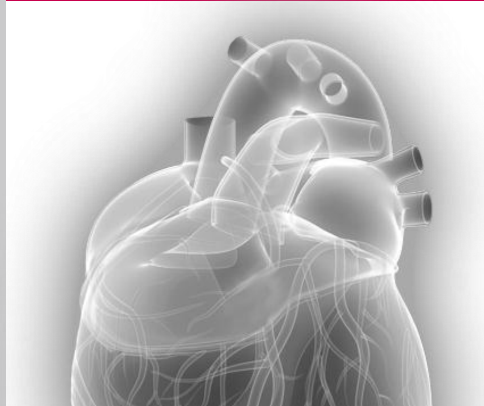


Content

Epidemiology



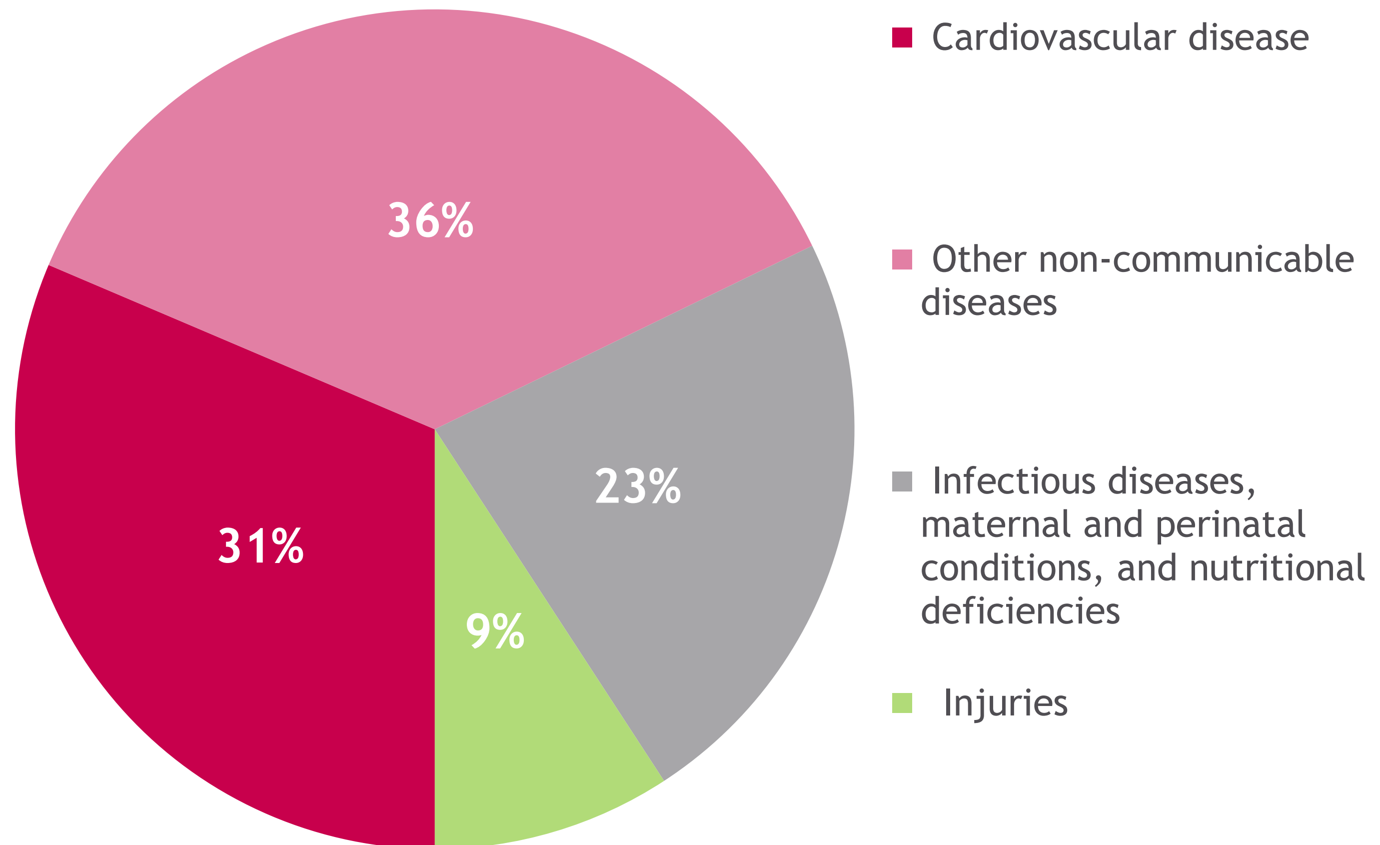
Pathophysiology



Symptoms & diagnosis



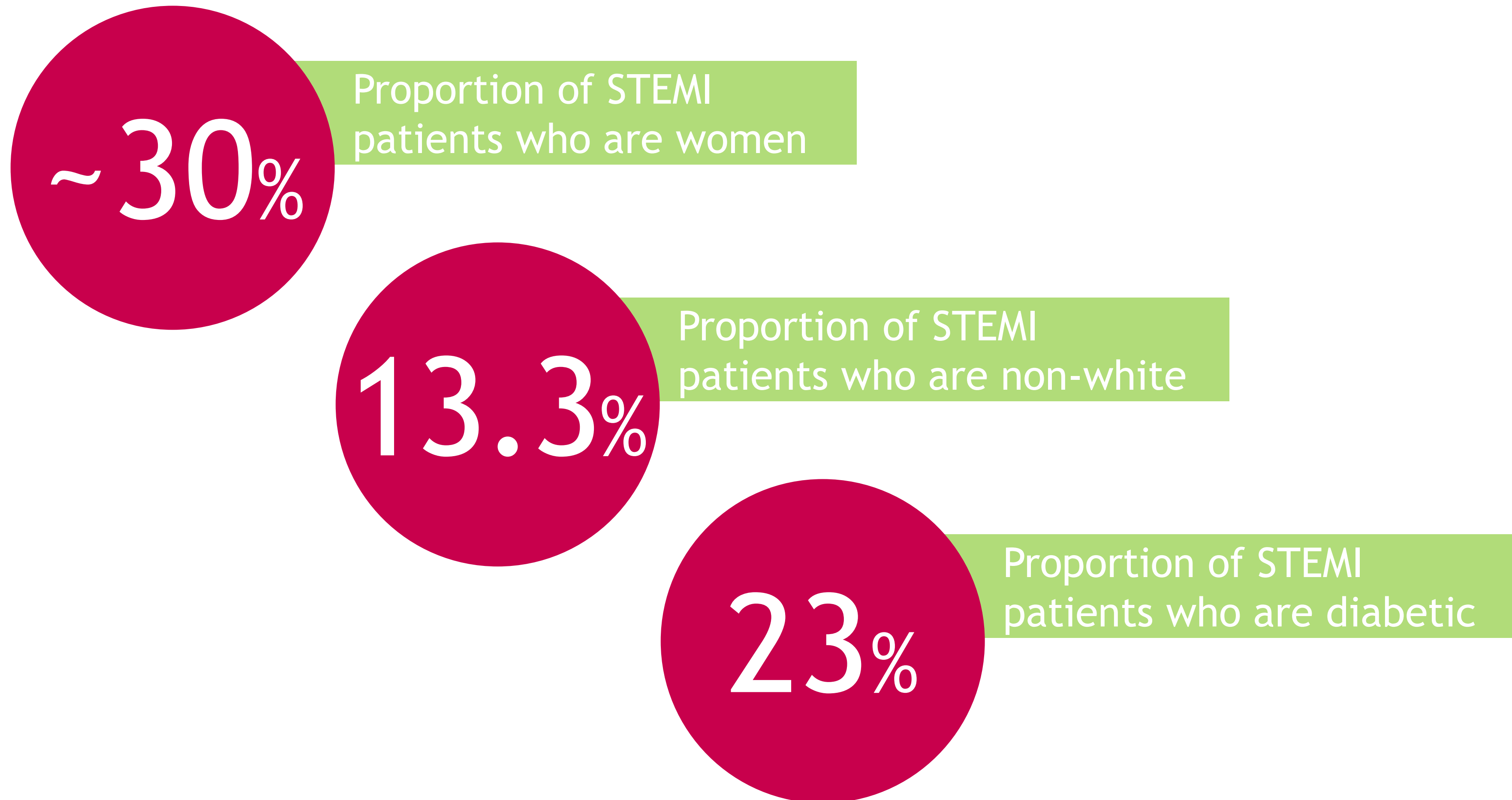
CVD is a major cause of death worldwide



Acute myocardial infarction (AMI): facts

- AMI is a leading cause of death worldwide[1]
- Approximately 42% of all deaths from cardiovascular disease are due to AMI[2]
- Men have a higher risk than women for AMI[3]
- Risk of AMI increases with advancing age for both genders[3]
- Black men & women are more at risk than white men & women[3]
- Incidence is increasing in developing and transitional countries, partly due to increasing longevity, urbanisation and lifestyle changes[3]

Populations based on data from the USA

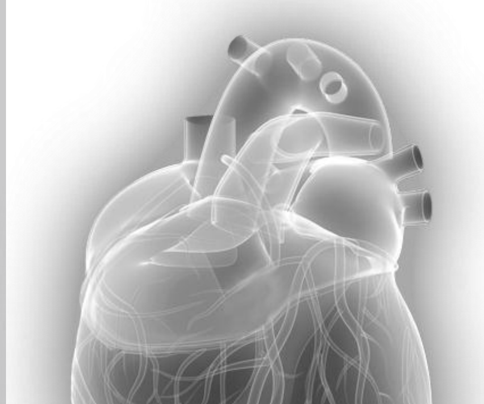


Pathophysiology

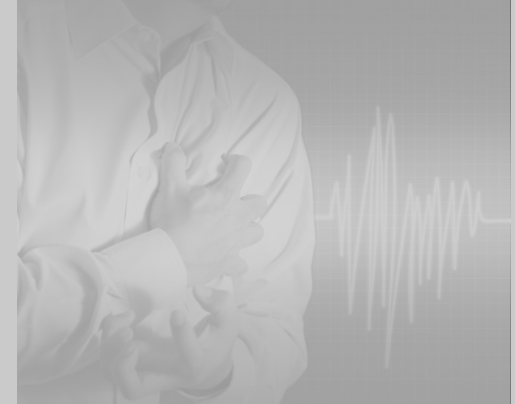
Epidemiology



Pathophysiology

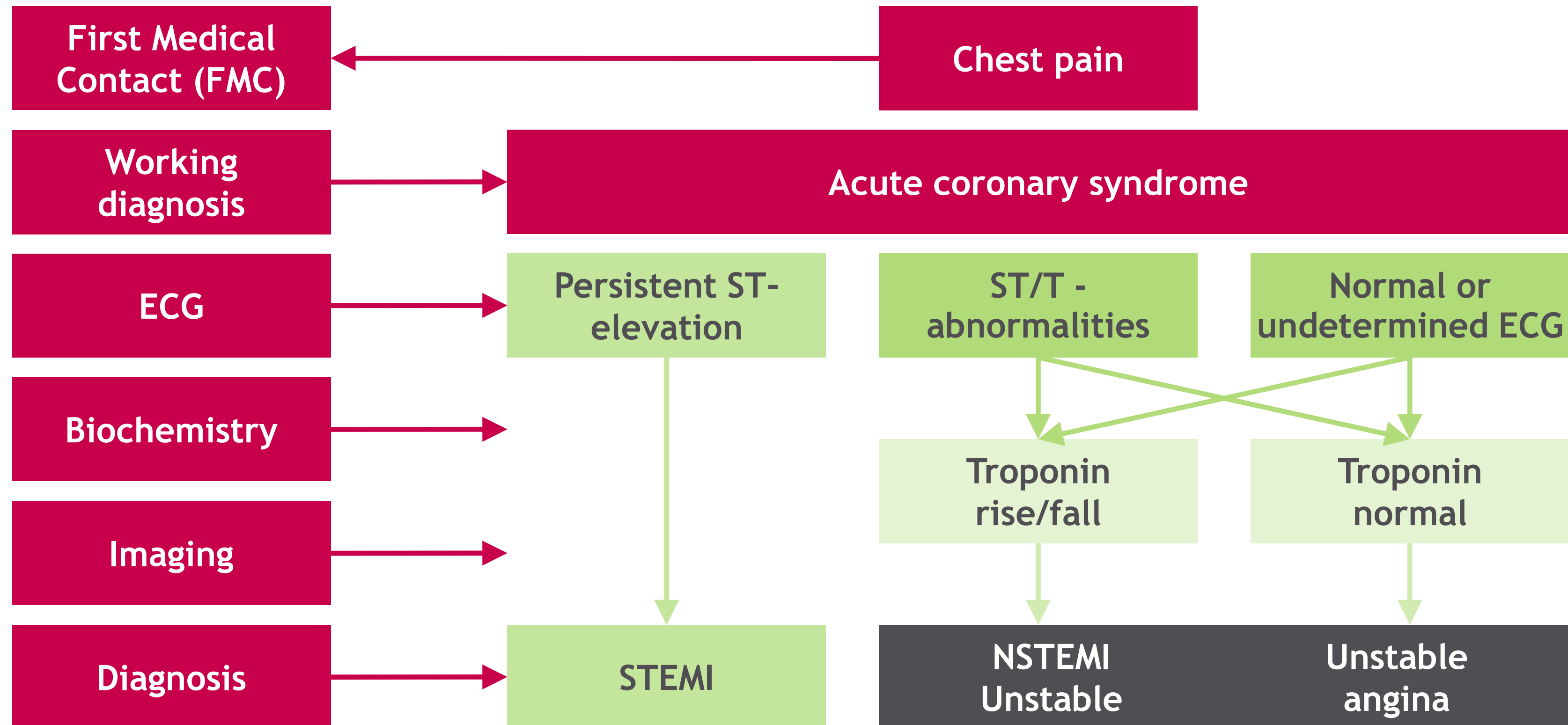


Symptoms & diagnosis

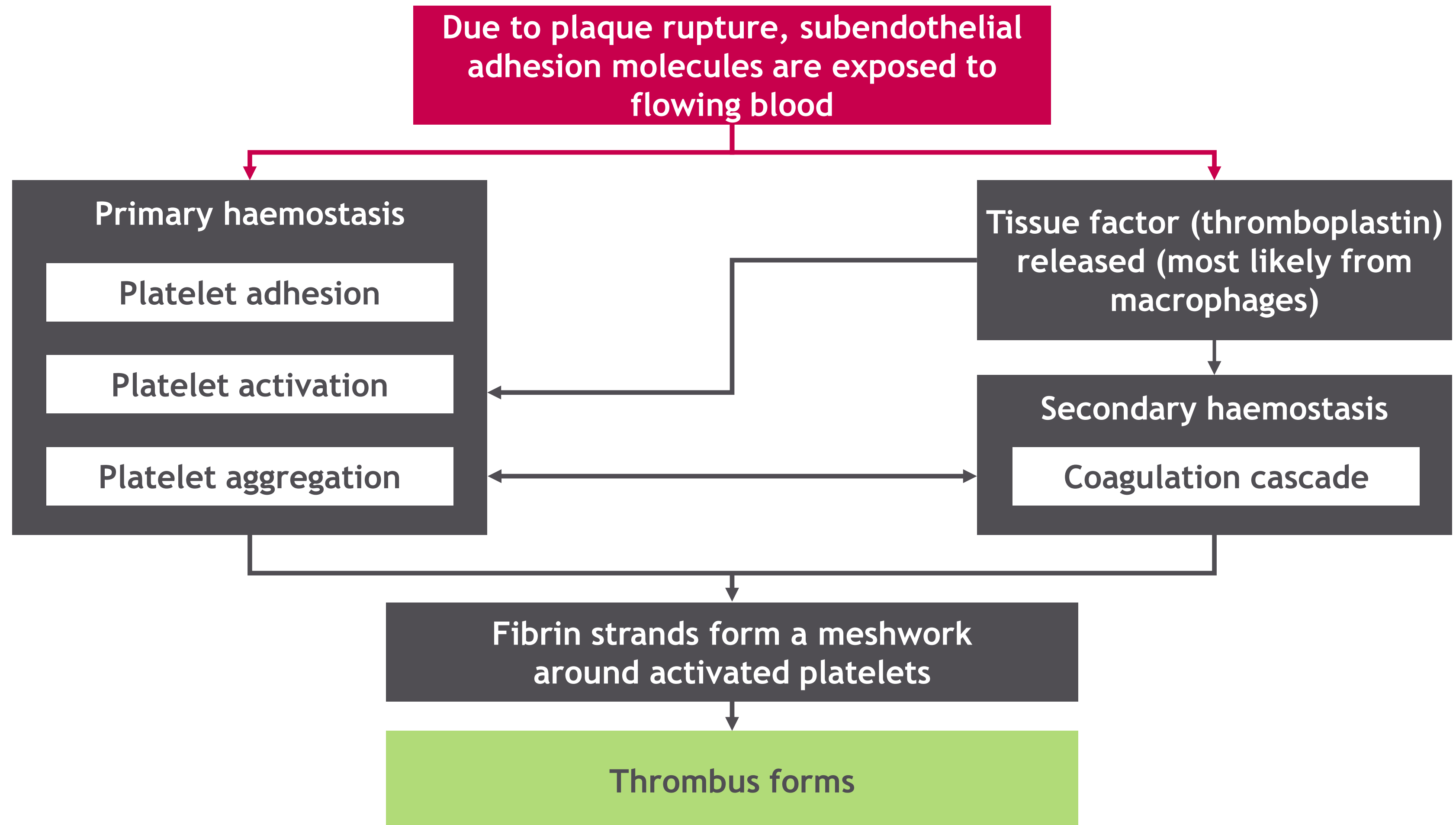


Acute coronary syndrome (ACS)

One disease process but different clinical manifestations and different management strategies

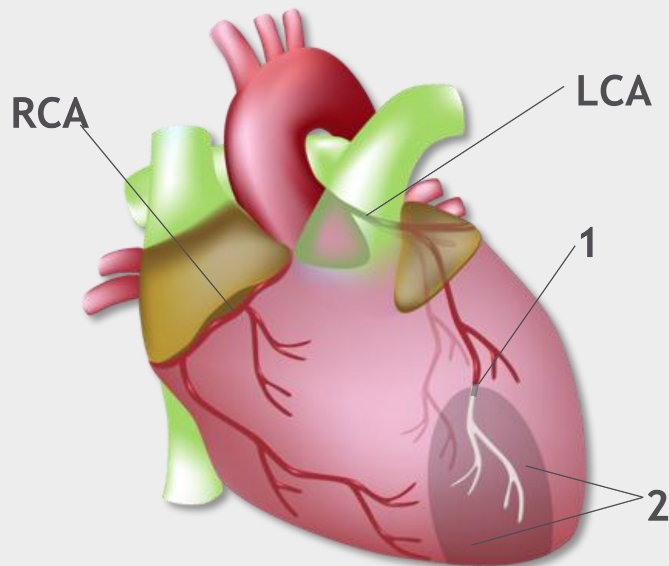


Thrombus formation in MI and other ACS



Definition of STEMI

A clinical syndrome defined by characteristic symptoms of myocardial ischaemia in association with persistent ECG ST-elevation and subsequent release of biomarkers of myocardial necrosis[1]



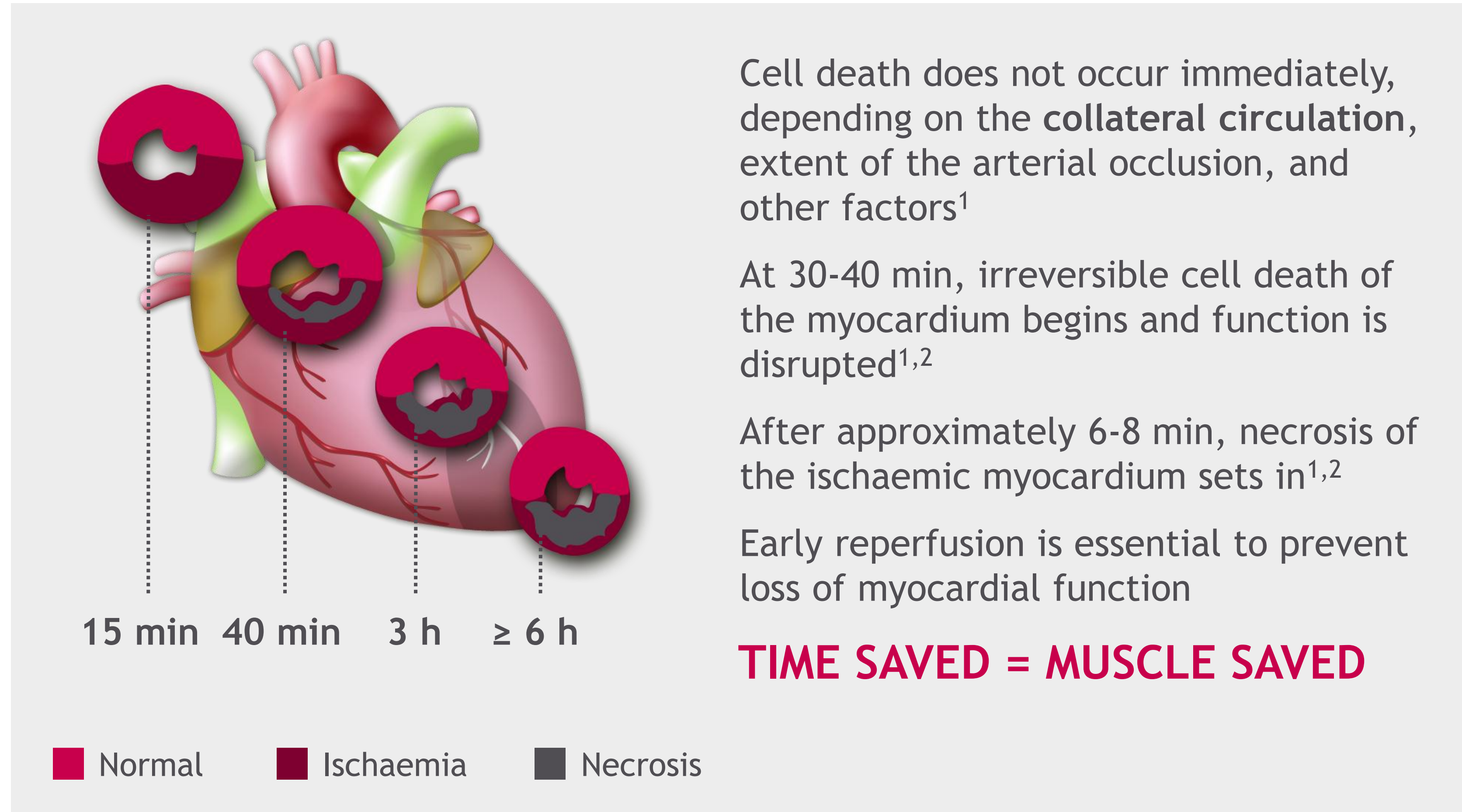
STEMI: full thickness damage (myocardial cell death) of cardiac muscle

NSTEMI: partial thickness damage of cardiac muscle

1 = obstructed artery
2 = infarcted area distal to the blocked artery

RCA, right coronary artery; LCA, left coronary artery; NSTEMI, non-ST-elevation myocardial infarction

Advancing necrosis in MI



Cell death does not occur immediately, depending on the **collateral circulation**, extent of the arterial occlusion, and other factors[1]

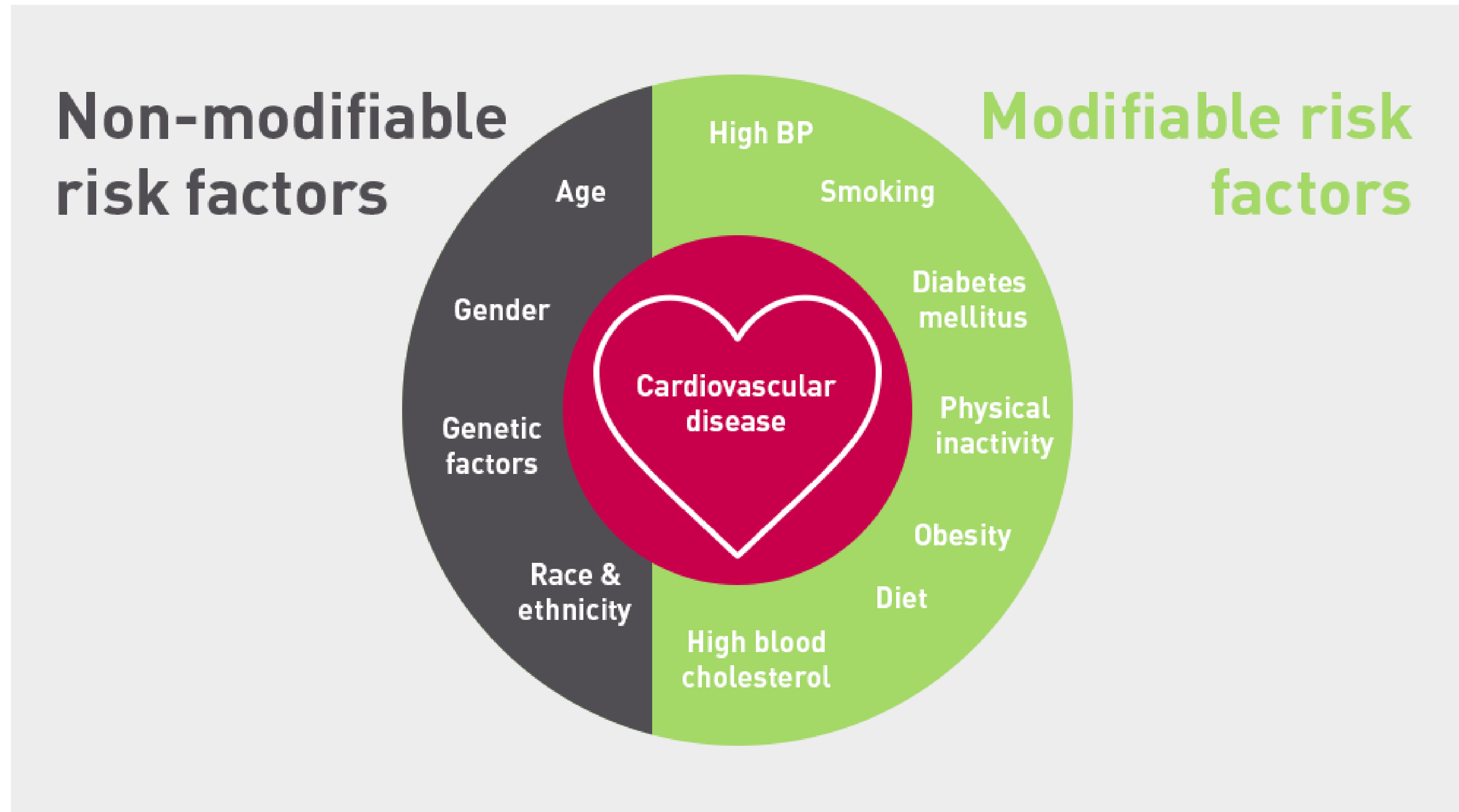
At 30-40 min, irreversible cell death of the myocardium begins and function is disrupted[1,2]

After approximately 6-8 min, necrosis of the ischaemic myocardium sets in[1,2]

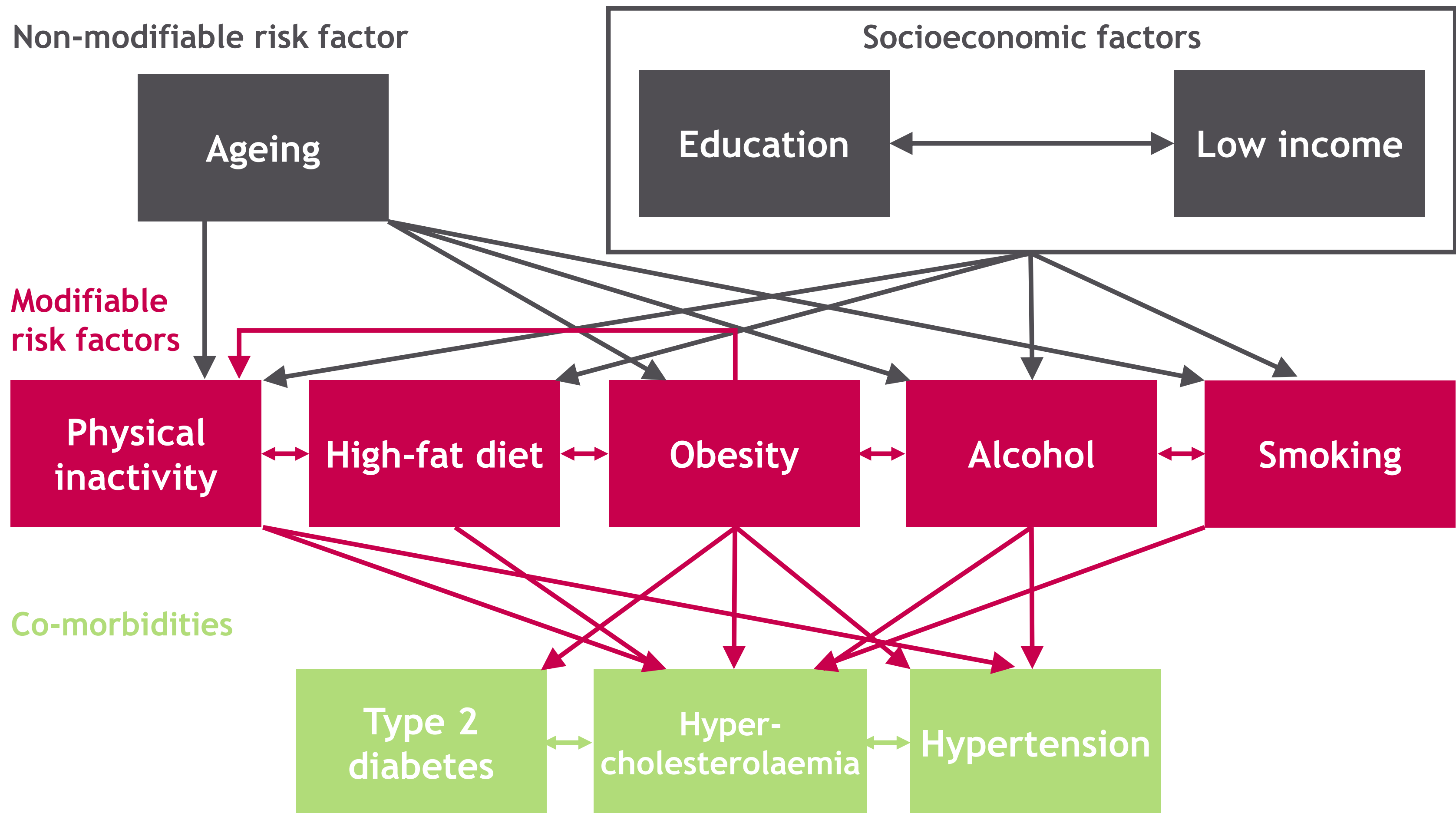
Early reperfusion is essential to prevent loss of myocardial function

TIME SAVED = MUSCLE SAVED

Major modifiable and non-modifiable risk factors for cardiovascular disease



Risk factors for IHD rarely occur alone



Other risk factors

Gender	• Higher rates of coronary heart disease among men compared with pre-menopausal women[1] • Risk for post-menopausal women is similar to men[1]
Heredity and family history	• Increased risk if first-degree blood relative had coronary heart disease or stroke[1] • Having a sibling with a history of CVD is associated with a 45% increased risk of CVD[3]
Summary	• In terms of attributable deaths, globally, CV risk factors are[2]: • Raised blood pressure (accounting for 13% of global deaths) • Tobacco use (9%) • Raised blood glucose (6%) • Physical inactivity (6%) • Overweight and obesity (5%)

Symptoms & diagnosis

Epidemiology



Pathophysiology



Symptoms & diagnosis



Typical symptoms of acute myocardial infarction (AMI)

- Onset may be sudden or gradual
- Symptoms vary depending on the location of the infarct

Chest pain or discomfort	Often described as a tightness, heaviness or constriction in the chest Usually in the centre of the chest, but radiate to neck, jaw, stomach, shoulder, back and arms (typically left arm)
Breathing difficulty / shortness of breath	Due to left ventricular dysfunction or dynamic mitral regurgitation
Profuse sweating	
Nausea and/or vomiting	
Dizziness	
Syncope	Usually due to an arrhythmia or severe hypotension
Tachycardia	Due to sympathetic nerve activation
Bradycardia	Patients with inferior STEMI may have bradycardia due to vagus nerve activation
Cardiogenic shock	Due to impaired myocardial function

Diagnostic criteria for AMI

Any one of the following criteria meets the diagnosis for AMI according to the Joint ESC/ACCF/AHA/WHF Task Force:

A rise and/or fall of cardiac biomarkers (preferably troponin (cTn)) with at least one value above the 99^{th} percentile upper reference limit (URL) together with at least one of the following:

+

- Symptoms of ischaemia
- New or presumed-new significant ST-segment-T wave (ST-T) changes or new left bundle branch block (LBBB)
- Development of pathological Q waves in the ECG
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
- Identification of an intracoronary thrombus by angiography or autopsy

Cardiac death with prior new ischaemic ECG changes and symptoms suggestive of myocardial ischaemia, without definitive biomarker evidence

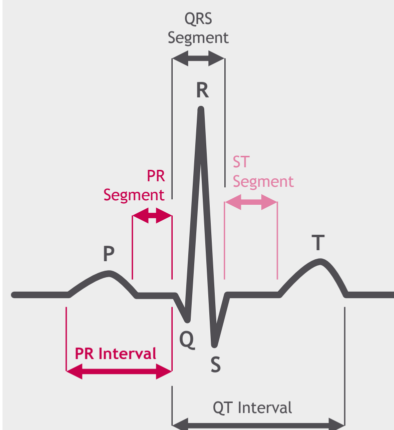
PCI-related MI*

Stent thrombosis associated with MI*

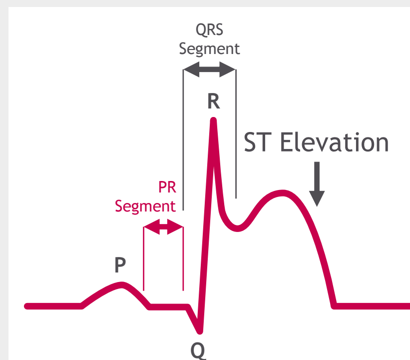
Coronary artery bypass grafting (CABG)-related MI*

Diagnosis of STEMI: ECG changes

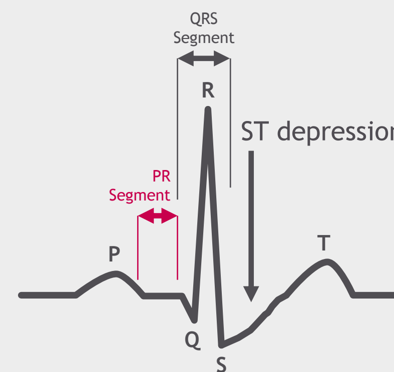
- ST-segment elevation with pathological Q-wave formation
- Sometimes T-wave inversion may be found but it is a non-specific feature
- ST-segment elevation indicates full thickness cardiac muscle injury, pathological Q-wave indicates muscle necrosis and T-wave inversion indicates muscle ischaemia



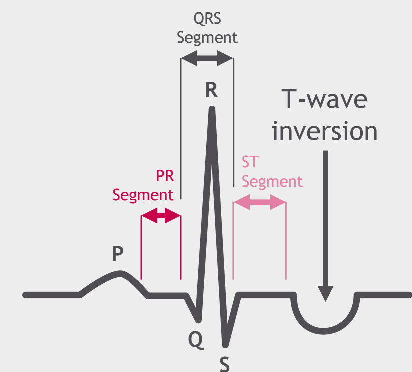
Normal ECG



STEMI



NSTEMI



NSTEMI

Diagnosis of STEMI

Cardiac markers
- Troponin is the preferred biomarker for diagnosis

Full blood count
- Elevation of white blood cell count is usual
- Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) may be elevated

Chest X-ray
- For assessing pulmonary oedema

Echocardiography
- Not essential, but helpful if ECG is inconclusive

STEMI types defined by ECG changes

STEMI type	Area affected	Occluded vessel	ECG findings		Prognosis
			ST segment elevation	Reciprocal ST-segment depression	
Anterior	Anterior wall of LV	LAD branch of LCA	Leads V1 - V6	Inferior leads II, III and aVF	Poor
Antero-septal	Area between LV and RV	LAD septal branches	Leads V1 - V4	Inferior leads II, III and aVF)	--
Lateral	Lateral wall of LV	1st diagonal branch of LAD and obtuse marginal branch of LCX	Leads I, aVL, V5 and V6	Inferior leads II, III and aVF	--
High lateral	Superior portion of the lateral wall of LV	1st diagonal branch of LAD	Leads I and aVL	Inferior leads II, III and aVF	--
Antero-lateral	Anterior and lateral wall of LV	Proximal LAD or LAD + LCX	Leads I, aVL, V4 - V6	Inferior leads II, III and aVF	--
Inferior	Inferior wall of LV	RCA	Leads II, III and aVF	Leads I and aVL	Good[a]
Posterior	Posterior part of LV	Posterior descending artery	Leads V7 - V9 (posterior leads)[b]		--
RV infarction[c]			Right sided chest leads (V3 R– V6 R).		--

TREATMENT

- A. Aspirin, P2Y12 Inhibitors (Prasugrel, Ticagrelor, and Clopidogrel)

- All patients with definite or suspected acute MI should receive aspirin at a dose of 162 mg or 325 mg at once regardless of whether fibrinolytic therapy is being considered or the patient has been taking aspirin. Chewable aspirin provides more rapid blood levels. Patients with a definite aspirin allergy should be treated with a P2Y12 inhibitor (clopidogrel, prasugrel, or ticagrelor).
- P2Y12 inhibitors, in combination with aspirin, have been shown to provide important benefits in patients with acute STEMI. Thus, guidelines call for a P2Y12 inhibitor to be added to aspirin for all patients with STEMI, regardless of whether reperfusion is given, and continued for at least 14 days, and generally for 1 year

B. Reperfusion Therapy

- Patients with STEMI who seek medical attention within 12 hours of the onset of
- symptoms should be treated with reperfusion therapy, either primary PCI or fibrinolytic
- therapy. Patients without ST-segment elevation (previously labeled “non-Q wave”
- infarctions) do not benefit, and may derive harm, from thrombolysis.

1. Primary percutaneous coronary intervention

2. Fibrinolytic therapy

- A. BENEFIT—Fibrinolytic therapy reduces mortality and limits infarct size in patients with STEMI (defined as 0.1 mV or more in two inferior or lateral leads or two contiguous precordial leads), or with left bundle branch block (not known to be old).

- the greatest benefit occurs if treatment is initiated within the first 3 hours after the onset of presentation, when up to a 50% reduction in mortality rate can be achieved. The magnitude of benefit declines rapidly thereafter, but a 10% relative mortality reduction can be achieved up to 12 hours after the onset of chest pain.

- The survival benefit is greatest in patients with large—usually anterior—infarctions. Primary PCI (including stenting) of the infarct-related artery, however, is superior to thrombolysis when done by experienced operators with rapid time from first medical contact to intervention ("door-to-balloon")

B. CONTRAINDICATIONS

C. FIBRINOLYTIC AGENTS

- Alteplase
- Reteplase
- Tenecteplase
- Streptokinase

(1) Selection of a fibrinolytic agent—

(2) Postfibrinolytic management

- (A) LOW-MOLECULAR-WEIGHT HEPARIN—
- (B) UNFRACTIONATED HEPARIN
- (C) PROPHYLACTIC THERAPY AGAINST GASTROINTESTINAL BLEEDING

3. Assessment of myocardial reperfusion, recurrent ischemic pain, reinfarction—

- Myocardial reperfusion can be recognized clinically by the early cessation of pain and the resolution of ST-segment elevation. Although at least 50% resolution of ST-segment
- vessels will reocclude during hospitalization, although reocclusion and reinfarction appear to be reduced following intervention. Reinfarction, indicated by recurrence of pain and ST-segment elevation, can be treated by readministration of a thrombolytic
- agent or immediate angiography and PCI.

C. General Measures

- Cardiac care unit monitoring should be instituted as soon as possible. Patients without
- complications can be transferred to a telemetry unit after 24 hours. Activity should
- initially be limited to bed rest but can be advanced within 24 hours. Progressive
- ambulation should be started after 24–72 hours if tolerated. For patients without
- complications, discharge by day 4 appears to be appropriate. Low-flow oxygen therapy
- (2–4 L/min) should be given if oxygen saturation is reduced, but there is no value to
- routine use of oxygen.

D. Analgesia

- An initial attempt should be made to relieve pain with sublingual nitroglycerin.
- However, if no response occurs after two or three tablets, intravenous opioids provide
- the most rapid and effective analgesia and may also reduce pulmonary congestion.
- Morphine sulfate, 4–8 mg, or meperidine, 50–75 mg, should be given. Subsequent small
- doses can be given every 15 minutes until pain abates.
- Nonsteroidal anti-inflammatory agents, other than aspirin, should be avoided during
- hospitalization for STEMI due to increased risk of mortality, myocardial rupture,

E. Beta-Adrenergic Blocking Agents

- Trials have shown modest short-term benefit from beta-blockers started during the first
- 24 hours after acute MI if there are no contraindications (metoprolol 25–50 mg orally
- twice daily). Aggressive beta-blockade can increase shock, with overall harm in
- patients with heart failure. Thus, early beta-blockade should be avoided in patients with
- any degree of heart failure, evidence of low output state, increased risk of cardiogenic
- shock, or other relative contraindications to beta-blockade. Carvedilol (beginning at
- 6.25 mg twice a day, titrated to 25 mg twice a day) was shown to be beneficial in the

E. Beta-Adrenergic Blocking Agents

- CAPRICORN trial following the acute phase of large MI

F. Nitrates

- Nitroglycerin is the agent of choice for continued or recurrent ischemic pain and is
- useful in lowering BP or relieving pulmonary congestion. However, routine nitrate
- administration is not recommended, since no improvement in outcome has been

observed in the ISIS-4 or GISSI-3 trials. Nitrates should be avoided in patients who

- received phosphodiesterase inhibitors (sildenafil, vardenafil, and tadalafil) in the prior

24 hours

G. Angiotensin-Converting Enzyme (ACE) Inhibitors

- A series of trials (SAVE, AIRE, SMILE, TRACE, GISSI-III, and ISIS-4) have shown
- both short- and long-term improvement in survival with ACE inhibitor therapy. The
- benefits are greatest in patients with an EF of 40% or less, large infarctions, or clinical
- evidence of heart failure. Because substantial amounts of the survival benefit occur on
- the first day, ACE inhibitor treatment should be commenced early in patients without
- hypotension, especially patients with large or anterior MI. Given the benefits of ACE
- inhibitors for patients with vascular disease, it is reasonable to use ACE inhibitors for
- all patients following STEMI who do not have contraindications.

H. Angiotensin Receptor Blockers

- Although there has been inconsistency in the effects of different ARBs on mortality for
- patients post-MI with heart failure and/or LV dysfunction, the VALIANT trial showed
- that valsartan 160 mg orally twice a day is equivalent to captopril in reducing mortality.
- Thus, valsartan should be used for all patients with ACE inhibitor intolerance, and is a
- reasonable, albeit more expensive, alternative to captopril. The combination of
- captopril and valsartan (at a reduced dose) was no better than either agent alone and
- resulted in more side effects.

I. Aldosterone Antagonists

- The RALES trial showed that 25-mg spironolactone can reduce the mortality rate of
- patients with advanced heart failure, and the EPHESUS trial showed a 15% relative
- risk reduction in mortality with eplerenone 25 mg daily for patients post-MI with LV
- dysfunction (LVEF of 40% or less) and either clinical heart failure or diabetes. Kidney
- dysfunction or hyperkalemia are contraindications, and patients must be monitored
- carefully for development of hyperkalemia

J. Calcium Channel Blockers

- There are no studies to support the routine use of calcium channel blockers in most
- patients with acute MI—and indeed, they have the potential to exacerbate ischemia and
- cause death from reflex tachycardia or myocardial depression. Long-acting calcium
- channel blockers should generally be reserved for management of hypertension or
- ischemia as second- or third-line medications after beta-blockers and nitrates.

K. Long-Term Antithrombotic Therapy

L. Coronary Angiography

- For patients who do not reperfuse based on lack of at least 50% resolution of ST elevation, rescue angioplasty should be performed and has been shown to reduce the composite risk of death, reinfarction, stroke, or severe heart failure. Patients treated

- with coronary angiography and PCI 3–24 hours after fibrinolytic therapy showed improved outcomes. Patients with recurrent ischemic pain prior to discharge should undergo catheterization and, if indicated, revascularization. PCI of a totally occluded

- infarct-related artery more than 24 hours after STEMI should generally not be performed in asymptomatic patients with one or two vessel disease without evidence of
- severe ischemia

Associat Prof. Mohammed ghaleb

